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**Influence of Low and High Pressure Baroreceptors
on Plasma Renin Activity in Humans**

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Abstract

We evaluated effects of low and high pressure baroreceptors on plasma renin activity (immunoassay) using graded lower body suction (LBS) in six healthy men. LBS at -10 and -20 mmHg for 10 min decreased central venous pressure without changing arterial pressure and thereby presumably reduced low but not high pressure baroreceptor inhibition of renin release. LBS at these levels produced forearm vasoconstriction, but did not increase renin. LBS at -40 mmHg decreased central venous and arterial pulse pressure and thus reduced both low and high pressure baroreceptor inhibition. LBS at this level produced forearm vasoconstriction and tachycardia and increased renin from 2.1 ± 0.4 (mean \pm SE) to 7.4 ± 1.4 ng \cdot ml $^{-1}$ \cdot hr $^{-1}$ ($p < 0.05$). In summary, reduction in low pressure baroreceptor inhibition in humans did not increase renin in the presence of physiological tonic inhibition from high pressure baroreceptors. Increases in renin did not occur until there was combined reduction of high and low pressure baroreceptor inhibition on plasma renin activity.

Index Terms

Cardiopulmonary Baroreceptors
Arterial Baroreceptors
Forearm Blood Flow (Plethysmography)

Decreases in baroreceptor restraint during upright posture (4,8,23) or lower body suction (11,12) increase plasma renin activity. These increases in renin activity play an essential role in maintenance of blood pressure during upright posture in humans during low sodium intake (24).

Several studies have evaluated the reflex mechanisms involved in renin release. Experiments in animals suggest that low pressure (cardiopulmonary) baroreceptors may participate in reflex regulation of plasma renin activity (3,18,20, 28,29,32).

However, the role of high pressure (carotid and aortic) baroreceptors in reflex regulation of renin activity is unclear. Several investigators (7,17,29) have reported that sinoaortic denervation or bilateral carotid occlusion increases renin. Other investigators (2,20,25) have not observed an increase in renin with decreases in carotid sinus pressure.

Jarecki, Thames and Donald (18) recently demonstrated an interaction between cardiopulmonary and carotid baroreceptors in control of renin activity in dogs. These investigators reported that interrupting vagal afferent traffic from cardiopulmonary receptors increases renin when carotid baroreceptors are denervated, but not when carotid baroreceptors are intact. These findings are compatible with the concept that low and high pressure baroreceptors may both participate in the reflex control of renin activity in animals.

In this study we have employed lower body suction to examine baroreceptor control of plasma renin activity in humans. Lower body suction is considered to produce reflex circulatory adjustments primarily by influencing low and high pressure baroreceptors although a contribution of other reflexes has not been excluded in humans.

Low pressure (cardiopulmonary) baroreceptor restraint on renin release was reduced with low levels (-10 and -20 mmHg) of lower body suction (18,31). Lower body suction at -10 and -20 mmHg decreases central venous pressure without decreasing arterial pressure or high pressure baroreceptor inhibitory input. At higher levels of lower body suction (-40 mmHg) both central venous pressure and systemic arterial pulse pressure decrease so that restraint on renin from both low and high pressure baroreceptor is decreased.

Methods and Design

Six healthy young men, age 20-25 years, were studied supine in a warm room (26-27°C). The study was performed with the approval of the University of Iowa Committee on Research Involving Humans and with the informed, written consent of each subject. Forearm blood flow was measured in the right arm by venous occlusion plethysmography using a mercury-in-silastic strain gauge (1,14,33). Heart rate was obtained from an electrocardiogram. Systemic blood pressure was measured with a sphygmomanometer. Using local anesthesia, a polyethylene cannula (60 cm long; I.D. 0.7 mm) was inserted into an antecubital vein and advanced into an intrathoracic vein to measure central venous pressure and to sample blood for determination of plasma renin activity.

Plasma renin activity was determined by radioimmunoassay by modification of the method of Haber et al (15). Seven ml of blood were collected in a chilled EDTA tube. The samples were centrifuged in the cold at 4°C for 10 min. Plasma was obtained and frozen until assayed. Angiotensin I (¹²⁵I) Radioimmunoassay Kit (New England Nuclear) was used, with modification of plasma prior to incubation to pH 5.5. One half of each sample was incubated at 1 hour at 37°C, the remainder was incubated at 4°C. A duplicate standard curve

was performed for each immuneassay. All samples from a given experiment were run simultaneously, and all were run within two weeks of the experiment. All experimental plasmas were also run in duplicate.

Renin activity was determined by subtracting the angiotensin I present in the 4°C sample from that obtained in the sample incubated at 37°C for one hour. Results are recorded as $\text{ng}\cdot\text{ml}^{-1}\cdot\text{hr}^{-1}$. The mean variance of 29 duplicate pairs for this method using split samples from normal, high and low subjects done on two different days is 0.21.

Lower body suction (application of subatmospheric pressure) was produced by enclosing the lower part of the subject's body caudal to the iliac crests in an airtight chamber (33). A commercial vacuum cleaner was modified with a rheostat and connected to the chamber to produce graded lower body suction. The subjects had been exposed to lower body suction in previous experiments and tolerated suction without adverse effects.'

After experimental preparation, the subjects rested quietly for 20 min before we began the protocol. We then obtained responses to lower body suction at -10, -20 and -40 mmHg for 10 min each. Each intervention was separated by 10-20 min. The order of interventions was varied and was not known to

the individual performing the renin determinations. Plasma renin activity was measured in control periods, the 10th min of suction, and recovery periods. In two subjects, responses to LBS -20 mmHg were not obtained during the first session and were obtained during a separate session.

In four subjects, we obtained responses to lower body suction at -10 mmHg for 20 min to determine if more prolonged decreased in low pressure baroreceptor restraint might produce significant increases in renin.

In three subjects, responses to LBS -40 mmHg were obtained after intravenous administration of propranolol, $0.15 \text{ mg} \cdot \text{kg}^{-1}$. This was performed to demonstrate that the increases in renin were mediated through activation of beta adrenergic receptors and not from decreases in clearance of renin or from decreases in pulse pressure acting via the renal vascular stretch receptor.

Three subjects were restudied after 3 days of a low sodium diet. The subjects were fed the low sodium diet in the Clinical Research Center as a liquid formula which contained 10 meq Na^+ /24 hr and 100 meq K^+ /24 hr. It might be noted here that strict adherence to the diet was not essential for the study and was, therefore, not assessed. However, we have previously demonstrated excellent compliance with this diet in the Clinical Research Center. Most importantly, the

baseline plasma renin activity was 5 to 8 times higher in the three subjects during the low sodium intake than during ad lib sodium intake. In this session, we obtained responses to lower body suction at -10 and -40 mmHg for 10 min each.

In the studies with lower body suction we placed the suction chamber caudal to the iliac crests to minimize application of suction to the kidneys. Nevertheless, to assure that transmission of subatmospheric pressure to kidneys was not a factor in the results, we obtained response to leg suction in three additional subjects. The suction chamber was applied distal to the upper thighs. The level of leg suction (-50 mmHg) was selected as the level which produced a decrease in central venous pressure comparable to that produced by lower body suction at -10 to -20 mmHg.

Statistical comparisons were performed using the t test for paired data.

Results

Decrease in low pressure baroreceptor restraint.

Lower body suction at -10 mmHg for 10 min decreased central venous pressure and forearm blood flow, but did not alter arterial pulse pressure, arterial mean pressure, or heart rate (Table 1). LBS at this level did not increase plasma renin activity (Table 1 and Figure 1).

Lower body suction at -20 mmHg decreased central venous pressure and forearm blood flow (Table 1). LBS at this level tended to decrease pulse pressure and increase heart rate and renin, but these changes were not significant (Table 1 and Figure 1).

A longer period (20 min) of lower body suction at -10 mmHg also did not increase renin activity; renin averaged 1.7 ± 0.2 and $2.0 \pm 0.2 \text{ ng} \cdot \text{ml}^{-1} \cdot \text{hr}^{-1}$ before and after 20 min of lower body suction, respectively.

Combined decrease in low and high pressure baroreceptor restraint. Lower body suction at -40 mmHg decreased central venous pressure, arterial pulse pressure and forearm blood flow and increased heart rate (Table 1). This stimulus produced a two to three fold increase in renin (Table 1 and Figure 1). Propranolol blocked the increases in renin during LBS -40 mmHg (Figure 2).

Responses during low sodium intake. During low sodium intake, control values for renin averaged approximately eight times the control values during ad lib sodium intake (Figure 3). Renin increased during LBS -40 mmHg (Figure 3). Renin averaged 15.6 ± 1.9 in control, 41.0 ± 12.9 during LBS -40 and $17.6 \pm 1.6 \text{ ng} \cdot \text{ml}^{-1} \cdot \text{hr}^{-1}$ in recovery during low sodium intake. In contrast, with LBS -10 mmHg during low sodium intake renin decreased slightly in two subjects and increased modestly in one subject (Figure 3). Renin averaged 16.5 ± 2.5 in control, 16.9 ± 3.0 during LBS -10 and $15.6 \pm 1.9 \text{ ml} \cdot \text{min}^{-1} \cdot 100\text{ml}^{-1}$ during recovery.

Responses to leg suction. In three additional subjects, leg suction at -50 mmHg produced decreases ($p < 0.05$) in central venous pressure and forearm blood flow which approximated those during lower body suction at -20 mmHg. Central venous pressure averaged 4.2 ± 0.2 in control, 0.3 ± 0.9 during leg suction, and 4.0 ± 1.0 mmHg during recovery. Forearm blood flow averaged 5.9 ± 1.2 in control, 4.4 ± 0.7 during leg suction, and 5.5 ± 0.6 during recovery. However, this stimulus, like LBS -10 and -20 mmHg, did not increase renin. Renin averaged 4.3 ± 2.3 in control, 4.6 ± 2.4 during leg suction, and 4.3 ± 1.9 during recovery.

Discussion

In this study, levels of venous pooling which decrease low but not high pressure baroreceptor restraint on renin release (LBS -10 and -20 mmHg) did not significantly increase plasma renin. Levels of venous pooling which produce combined decrease in high and low pressure baroreceptor restraint were required to increase plasma renin.

Decrease in low pressure baroreceptor restraint. We conclude that decrease of low pressure baroreceptor restraint does not significantly increase renin in the presence of normal tonic restraint or buffering from arterial baroreceptors. However, we should consider alternative explanations for the failure of LBS -10 and -20 mmHg to increase renin.

First, did these levels of lower body suction produce sufficient venous pooling to decrease low pressure baroreceptor inhibitory activity. In this and previous studies (19,33), LBS -10 and -20 mmHg decreased central venous pressure and triggered reflex forearm vasoconstriction in the absence of decreases in arterial pulse pressure or mean pressure. The likely source for these forearm vasomotor changes during LBS -10 and -20 mmHg is a decrease in low pressure baroreceptor restraint. However, we cannot from this or earlier studies (18,24,31) exclude a contribution of other reflexes originating in somatic or visceral receptors.

Second, was the renin system responsive to reflex stimuli? Decreases in baroreceptor restraint during LBS -40 mmHg produced significant increases in renin during both ad lib and low sodium diets. Thus, despite responsiveness of the renin system, selective decrease in low pressure baroreceptor restraint with LBS -10 and -20 mmHg did not significantly increase renin.

Lower body suction at -20 mmHg pools approximately 400-600 ml of blood (22). Several investigators have reported that removal of 400-600 ml of blood (approximately 10% of blood volume) does not increase renin (5,6,13,15). Failure of renin to increase with these levels of hemorrhage might be attributed to failure to inhibit low pressure baroreceptor restraint. However, the marked reduction in atrial or central venous pressures would suggest that there is inhibition of low pressure baroreceptors with these levels of hemorrhage (15). Furthermore, the finding in the present study that LBS -10 and -20 mmHg produces significant forearm vasoconstriction also suggests that these levels of venous pooling or hemorrhage are sufficient to decrease low pressure baroreceptor restraint.

Combined decrease in low and high pressure baroreceptor restraint. LBS -40 mmHg, which decreases arterial pulse pressure and reduces high as well as low pressure restraint, produced

striking increases in renin. Thus, whereas a selective decrease in low pressure restraint did not increase renin, combined decrease in low and high pressure baroreceptor restraint produced significant increases in renin.

Propranolol blocked the increases in renin during LBS -40 mmHg. This indicates that the increases resulted from stimulation of beta adrenergic receptors. We cannot from this study state that the increased renin resulted from neural as opposed to humoral stimulation of the renal beta receptor. However, Zanchetti (31) has demonstrated that the increase in renin during orthostatic stress in cats is mediated almost entirely by neural mechanism.

We should consider the possibility that influences other than baroreceptor reflexes triggered the increases in renin during LBS -40 mmHg. For example, decreases in cardiac output during LBS -40 mmHg might increase renin independent of baroreceptor reflexes by decreasing pulse pressure and stretch on the renovascular stretch receptor (9) or by decreasing splanchnic blood flow and hepatic clearance of renin (26). However, these mechanisms are unlikely since the increase in renin with LBS -40 mmHg was blocked by propranolol.

We cannot from these studies exclude a contribution of other reflexes originating in somatic and visceral receptors. However, it seems unlikely that reflexes originating in cutaneous, muscle or venous receptors in the lower extremities contributed because leg suction at -50 mmHg did not increase renin.

Increases in renin by LBS -40 mmHg, which decreased high and low pressure baroreceptor restraint, might be explained in three ways. One explanation might be that high pressure baroreceptors play the predominant role in the baroreceptor reflex control of renin in humans. However, Zanchetti (personal communication) has stated that selective inhibition of high pressure (carotid) baroreceptors produced by positive neck pressure does not increase renin in humans. Thus, it would appear that selective decrease of either low or high pressure baroreceptors restraint does not significantly increase renin in humans.

A second explanation might be that low and high pressure baroreceptors both exert a physiological tonic restraint on renin activity in humans. This could explain Zanchetti's observation that selective decrease of carotid baroreceptor inhibition does not increase renin since the presence of physiological restraint from low pressure baroreceptors would prevent increases in renin activity under these conditions.

This interpretation would also explain our finding that selective decrease in low pressure baroreceptor inhibition does not significantly increase renin in the presence of physiological restraint from high pressure baroreceptors. We suggest that reflex increases in plasma renin in humans result from a combined and not selective decrease in low and high pressure restraint. In other words, plasma renin increases when there is a concerted decrease in tonic restraint from low and high pressure baroreceptors during lower body suction -40 mmHg, but not when there is a selective decrease in restraint from low pressure baroreceptors during LBS -10 or -20 mmHg.

There is a third possible explanation for the finding that renin increases with LBS -40 mmHg, but not with LBS -10 and -20 mmHg. Decreases in central venous pressure are greater with LBS -40 mmHg (Δ CVP -5.8 mmHg) than with LBS -20 mmHg (Δ CVP -4.9 mmHg). Thus there is greater withdrawal of low pressure restraint during LBS -40 mmHg than there is during LBS -10 and -20 mmHg. If the threshold for decreases in central venous pressure required to trigger low pressure baroreceptor effects on renin are reached with lower body suction -40, but not with lower suction -10 and -20 mmHg then one might explain the increases in renin with LBS -40 mmHg on the basis of low pressure baroreceptors without implicating a role for high pressure baroreceptors. LaGrange et al (20), reported that neurogenically-mediated renin release is

separate from neurogenically-mediated vasoconstriction. In their experiments low levels of direct electrical stimulation of renal sympathetic nerves increased renin release with only minimal changes in vascular resistance. In our experiments, the stimulus of lower body suction -10 or -20 mmHg was sufficient to trigger low pressure effects on vascular resistance. Thus, based on the observations of LaGrange et al (20), one might have expected reflex increases in renin if low pressure baroreceptors exerted the dominant restraint on renin release.

In summary, decreases in low pressure baroreceptor restraint in humans did not significantly increase plasma renin activity in the presence of physiological tonic restraint from high pressure baroreceptors. Increases in plasma renin activity did not occur until there was combined decrease in low and high pressure baroreceptor inhibition. These results in humans support recent observations in animals (17) that high as well as low pressure baroreceptors participate in reflex regulation of renin activity.

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Figure Legends

- Figure 1. Changes in plasma renin activity during lower body suction (LBS) at -10, 20 and 40 mmHg. Entries are mean \pm SE for 6 subjects. Asterisk indicates $p < 0.05$
- Figure 2. Effects of lower body suction at -40 mmHg (LBS 40) on plasma renin activity before and after propranolol in three subjects.
- Figure 3. Effects of lower body suction at -10 mmHg (LBS 10) and -40 mmHg (LBS 40) on plasma renin activity during ad lib sodium intake and low sodium intake (10 meq/24 hrs) in three subjects.

Table 1
Responses to Lower Body Suction

	Control	LBS 10	Recovery	Control	LBS 20	Recovery	Control	LBS 40	Recovery
Central Venous Pressure (mmHg)	5.7±1.0	2.8±0.9**	5.2±0.8	6.4±0.8	1.5±1.0**	6.1±1.0	5.3±0.8	-0.5±0.9**	5.6±1.0
Arterial Mean Pressure (mmHg)	80.5±3.8	79.2±3.5	82.8±4.0	90.8±2.9	89.3±3.3	91.3±3.2	81.7±3.7	83.5±2.8	88.2±3.5
Arterial Pulse Pressure (mmHg)	55.3±1.5	52.3±2.8	53.0±3.1	43.8±4.1	40.0±5.0	46.5±2.9	54.6±3.8	32.0±2.6**	48.3±3.8
Forearm Blood Flow (ml·min ⁻¹ ·100 ml ⁻¹)	6.43±0.62	5.62±0.60**	6.25±0.61	5.32±0.57	4.33±0.45**	5.10±0.69	6.38±0.68	4.35±0.47**	5.53±0.52
Heart Rate (beats·min ⁻¹)	65.0±4.6	63.2±3.4	63.5±3.9	58.7±4.6	64.8±4.4	58.3±3.7	62.5±3.5	85.5±2.6**	57.3±3.9
Plasma Renin Activity (ng·ml ⁻¹ ·hr ⁻¹)	2.2±0.4	2.3±0.6	2.0±0.5	2.8±1.1	3.6±1.2	2.7±0.9	2.1±0.6	7.4±1.4**	3.9±0.9

Entries are mean ± SE for 6 subjects.

LBS = lower body suction at 10, 20, and 40 mmHg.

* p<0.05: LBS vs control values

** p<0.05: LBS vs average of control and recovery values.

In two subjects, responses to LBS 20 were not obtained in the first session and were obtained in a separate experimental session.

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Δ
Plasma Renin Activity
(ng \cdot ml⁻¹ \cdot hr⁻¹)

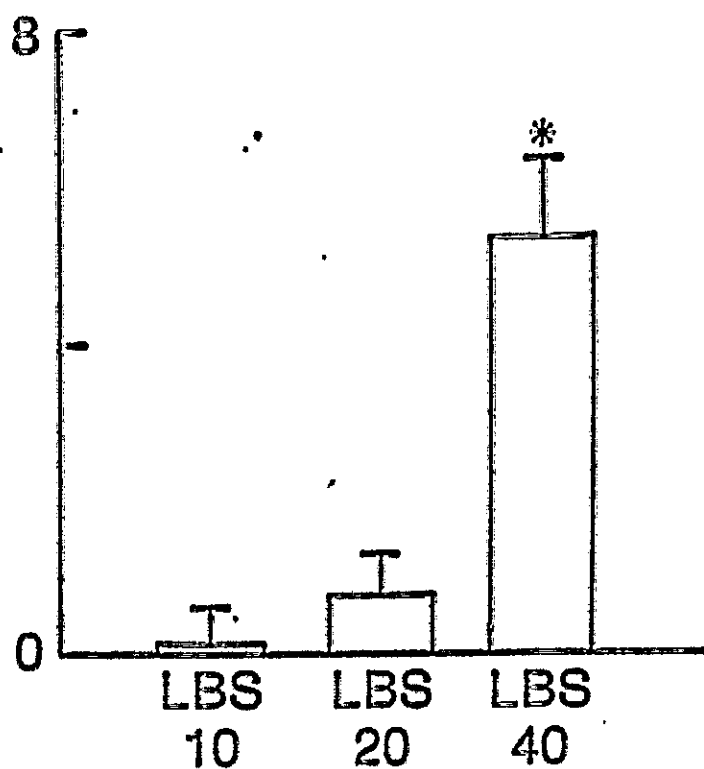


Figure 1

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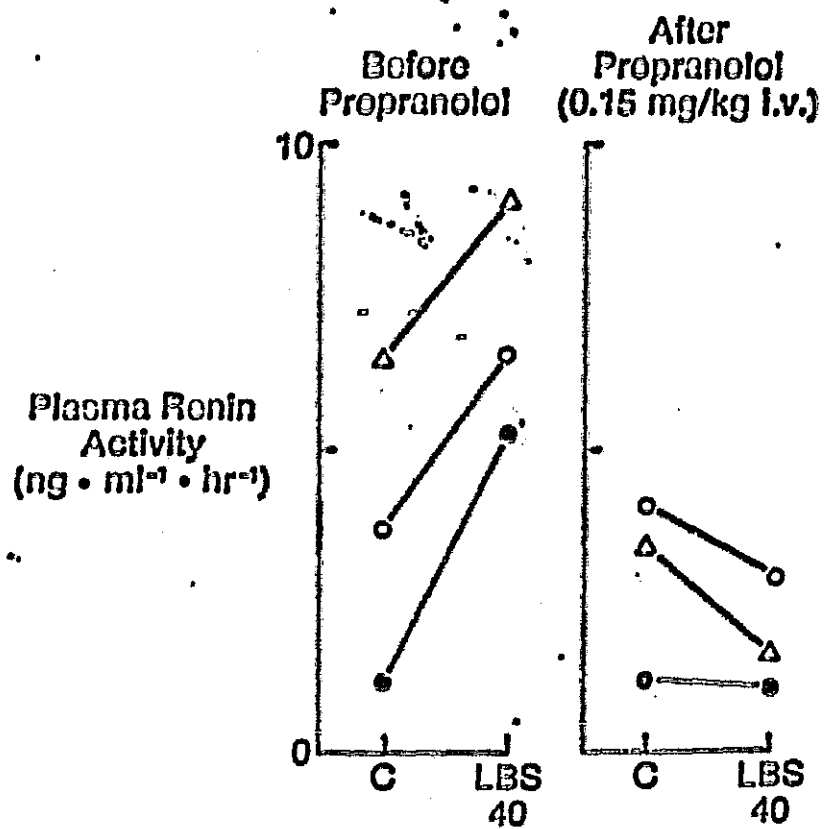


Figure 2

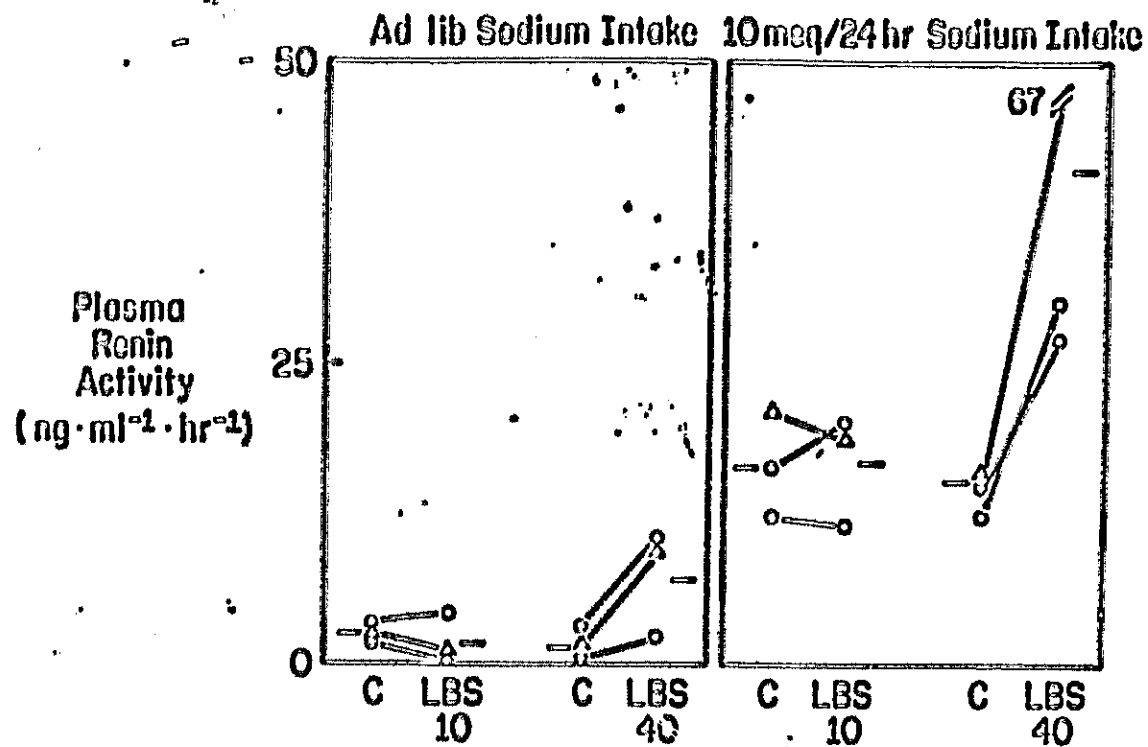


Figure 3

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